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Plant Health
Inspection
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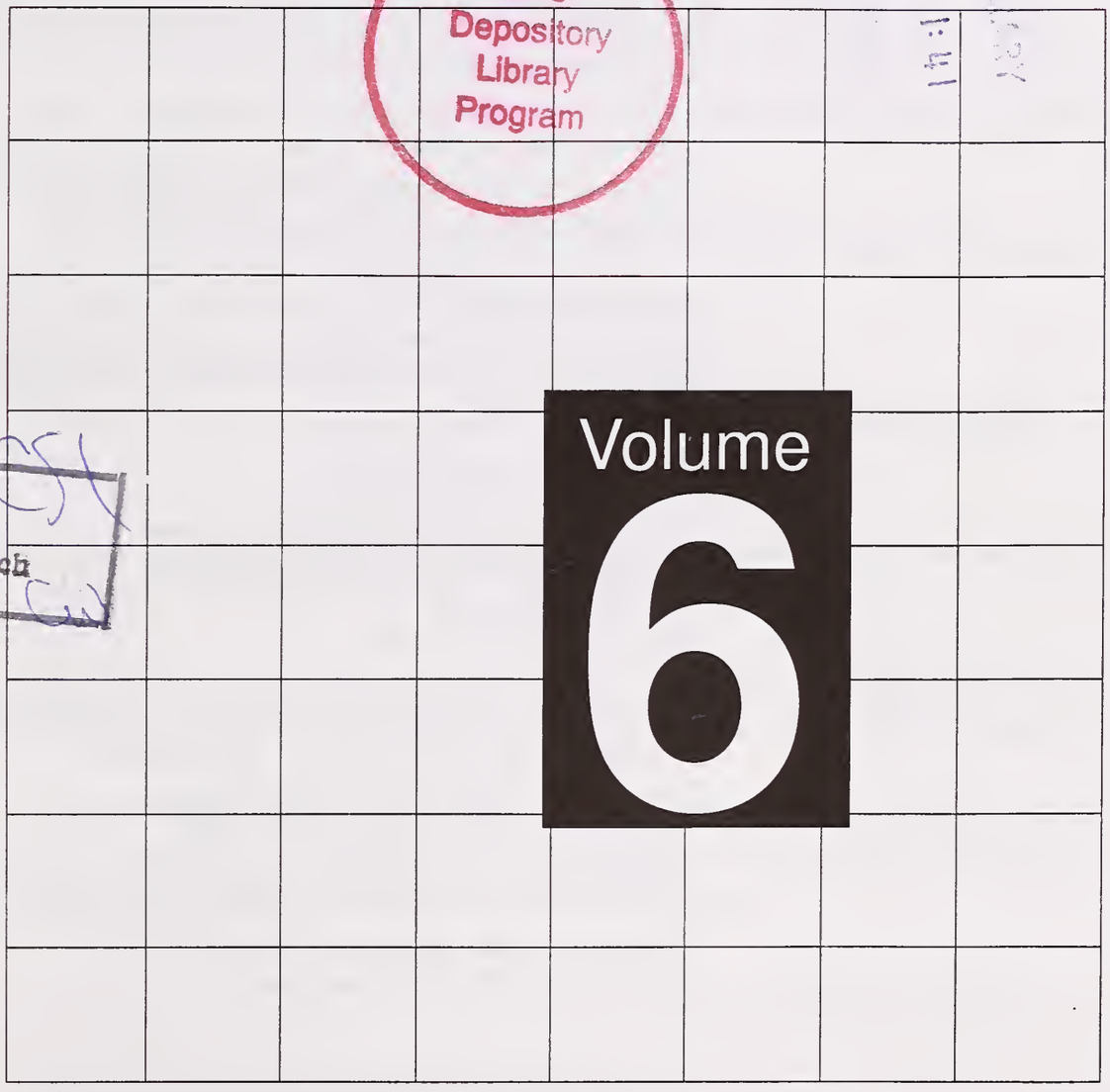
Program Aid 1581

Keeping America Free From Foreign Animal Diseases

Rinderpest, Peste des Petits Ruminants



Volume 6

Guidelines for Using This Package

This binder contains an integrated suite of educational materials about rinderpest and peste des petits ruminants. The package can be used in a formal training setting, where a presenter will show the video tape and narrate the slide show using this black-and-white brochure as the script. Or the materials can be used in a self-study program with the reader progressing at his or her own pace.

Within this brochure, readers will notice that certain paragraphs are preceded by a number. These numbers correlate to the slide set. For example, the rinderpest slides are all marked "RP" at the top of each plastic slide frame and numbered sequentially from 1 to 43. The peste des petits ruminants slides are marked "PPR" on top and numbered from 1 to 29.

If you remove the slides from their protective clear-plastic sleeve (for example, to put them into a carousel for group viewing), please be sure to reposition them in the correct numeric order for the benefit of future users.

This shrink-wrapped suite includes a video tape on rinderpest and peste des petits ruminants, two separate slide sets on these diseases, and the brochure you are reading now. If your package is incomplete, please contact the following office for replacement materials:

U.S. Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services, Emergency Programs
4700 River Road, Unit 41
Riverdale, MD 20737–1231

Instructional packages on other diseases are also available and may be requested by writing to the above address. Titles include
Program Aid 1576 African Horse Sickness
Program Aid 1577 African Swine Fever
Program Aid 1578 Contagious Bovine Pleuropneumonia
Program Aid 1579 Lumpy Skin Disease, Sheep Pox, Goat Pox
Program Aid 1580 Malignant Catarrhal Fever
Program Aid 1582 Vesicular Diseases

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Rinderpest

Definition

1 Rinderpest (RP) is a contagious viral disease of cattle, domestic buffalo, and wildlife that is characterized by fever, oral erosions, diarrhea, lymphoid necrosis, and high mortality.

Etiology

2 RP virus (RPV) is a single–stranded RNA virus in the family Paramyxoviridae, genus *Morbillivirus.* It is immunologically related to canine distemper virus, human measles virus, peste des petits ruminants virus, and marine mammal Morbilliviruses.

There is only one serotype of RPV, but field strains vary widely in virulence, ease of transmission, and host affinity.

RPV is a relatively fragile virus:

- Sunlight is lethal (vaccine must be in a brown bottle and protected from light; RPV in a thin layer of blood is inactivated in 2 hours).
- Moderate relative humidity inactivates the virus more quickly than either high or low humidity.
- The virus is very sensitive to heat. Both lyophilized and reconstituted virus should be kept cold; lyophilized virus stored at –20 °C is viable for years.
- Vaccine reconstituted in pure water quickly loses potency. The vaccine is more stable in a saline solution. Reconstitution in molar concentrations of sulfate ions will greatly increase resistance to heat.
- RPV is rapidly inactivated at pH 2 and 12 (10 minutes); optimal pH for survival is 6.5 to 7.
- RPV is inactivated by glycerol and lipid solvents.

Effective Disinfectants

3 Strong acid and alkaline disinfectants and many common disinfectants, particularly the iodophore and chlorine dioxide disinfectants, are effective.

History

4

Accounts of cattle plague go back as far as the siege of Troy in 1184 B.C. Since that time, RP has often been associated with war and movement of armies. Charlemagne brought RP to France when he returned with his armies in 810 A.D. From the 9th century to Napoleon (1800's), epizootics of RP swept over Europe every 50 years. RP was responsible for the establishment of the first veterinary school in Lyon, France, in 1762. During World War I, RP occurred in the Balkan countries, Russia and Poland. Even today, war is a factor, for during the war with Iraq in the early 1990's, RP entered Turkey when refugees from Iraq brought infected cattle with them.

1920: RP occurred in Belgium when infected cattle from India entered the port of Antwerp; 200 farms were infected.

1920: RP occurred in Brazil, probably introduced by Zebu cattle from India.

1923: RP occurred in Australia.

1969–73: Middle East pandemic. A focus of RP remained in Lebanon because of civil war, and the disease soon spread to Israel and Syria.

In North Africa, RP may have been present in what is now Senegal in the 1300's. Outbreaks of RP in North Africa up to 1865 tended to be self-limiting, but this changed with the "Great African Pandemic" of RP in 1880–95. In that pandemic, the RPV was believed to have been introduced by Indian cattle imported into Somalia in 1879. Eighty to 90 percent of the cattle and wild ruminants died in that pandemic—2.5 million cattle died in South Africa alone. The Masai tribes were reduced to starvation.

In Africa, 13 species of game animals are naturally affected by RP, and 6 more species can be infected experimentally. Of the game species, the buffalo and wildebeest are the greatest RP spreaders. Without reinfection from cattle, however, RP would probably die out in wild game animals. In the early 1900's, 2 million cattle and buffalo died of RP annually in Africa.

RP was nearly eliminated from Africa by the Joint Project No. 15 (JP–15) RP vaccination campaign from 1962 to 1976—a campaign sponsored by the United Nations and the Organization for African Union. Unfortunately, two small foci persisted because civil unrest in northeastern Africa meant that the animals could not be vaccinated. Then the program stopped, and most

countries did not continue vaccination because of its cost and local economic recession.

There was a great resurgence of RP in Africa from 1979 to 1983, and more than a million cattle died. From 1987 to the present, the Pan African Rinderpest Campaign (PARC), funded by the European Union and managed by the Organization for African Unity, has greatly reduced the incidence of RP there. The goal is to eradicate RP from Africa by the year 2000.

Host Range

5 Most wild and domestic cloven-footed animals can be infected.

Geographic Distribution

6 RP is present in the Indian subcontinent, the Near East, Egypt, and sub-Saharan Africa.

Transmission and Epidemiology

7 RP was established as an infectious disease in 1754, when susceptible animals were infected by placing bits of material previously dipped in morbid discharge into an incision made in the dewlap. In 1899, cattle were infected with bacteria-free filtrate.

Secretions and excretions, particularly nasal–ocular discharges and feces, from 1 to 2 days before clinical signs to 8 to 9 days after onset of clinical signs, contain large quantities of virus.

Spread of RP is by direct contact or indirect contact (contaminated ground, waters, equipment, clothing) with infected animals. Aerosol transmission of RP is not significant. It occurs only in a confined area and over a short distance. A major reason RP spreads in Africa is that the herds are nomadic. Cattle follow the grass and thus move great distances, and during the dry season, many herds will use the same well, providing ample opportunity for cross-infection. It is said colloquially that a good fence will control RP.

There is only one serotype of RPV; recovered and properly vaccinated animals are immune for life. Also, there is no vertical transmission, arthropod vector, or carrier state. For these reasons, RPV is an ideal virus to be targeted for eradication.

The highly virulent strains of RPV are responsible for epizootics in susceptible animals and tend to die out. The milder strains tend to persist in an area. The disease is not recognized as RP unless serological tests are performed.

8 Various hosts can play different roles in the disease:

- Cattle and domestic buffalo are highly susceptible.
- Sheep and goats in Africa may have subclinical infection and seroconversion but do not transmit RP.
- Sheep and goats in India, when infected by low passage goat RP vaccine, can transmit the disease to domestic buffalo.
- Pigs

—Swayback pigs in Thailand and the Malay peninsula can be naturally infected and may die.

—European pigs can be infected by ingestion of RPV-infected meat and can transmit the disease to cattle and other pigs.

- Wild ungulates

—Highly susceptible wild ungulates include the African buffalo, wildebeest, kudu, eland, giraffe, and warthog.

—Fairly susceptible wild ungulates include the Thompson's gazelle and hippopotamus.

Wild ungulates are infected by contact with cattle and can transmit RP to cattle. In the absence of cattle, the disease dies out.

Incubation Period

9 The incubation period varies with strain of RPV, dosage, and route of exposure. Following natural exposure, the incubation period ranges from 3 to 15 days but is usually 4 to 5 days.

Pathogenesis

10 Portal of entry is probably the epithelium of the upper respiratory tract and/or tonsil.

RPV replicates in the local lymph nodes. The virus spreads via the blood to other lymph nodes and the mucosa of the gastrointestinal and respiratory tracts. Because the virus replicates in lymphoid tissue and epithelium, wherever these two tissues occur together (e.g., in the Peyer's patches), the lesion is accentuated.

Destruction of the lymphoid tissue causes immunosuppression and may result in recrudescence of hemoparasitic and other secondary infections such as broncho-pneumonia.

When using animals to test for the presence of RPV, route of inoculation is important. Experimentally, using 1-mL aliquots from the same dilution of RPV, researchers found that animals inoculated in the jugular vein did not get sick while animals inoculated subcutaneously over the prescapular lymph node died of RP. A probable reason for the different results using these two routes of inoculation is that when the RPV was inoculated intravenously, the macrophages in the lung destroyed the virus; when inoculated subcutaneously, the virus went to the local lymph node and replicated.

Clinical Signs

11 Depending on the strain of virus, resistance of the animal affected and concurrent infection, RP can appear as a peracute, acute, or mild infection.

Of the Peracute Form of RP—seen in highly susceptible and young animals:

- No prodromal sign of illness
- High fever—104 to 107 °F (40 to 41.7 °C)
- Congested mucous membranes
- Death within 2 to 3 days after the onset of fever

12 **Of the Acute or Classic Form of RP**—This disease progresses as follows:

- Small amounts of virus may be in nasal and ocular secretions before the onset of fever.
- Fever of 104 to 106 °F (40 to 41.1 °C)
- Serous oculonasal discharge
- Leukopenia
- Depression
- Anorexia
- Constipation
- Oral erosions (salivation may be abundant and frothy)
- Decreases in fever and viral titer
- Diarrhea (may be very watery or hemorrhagic)
- Dehydration, emaciation
- Prostration and death 6 to 12 days after onset of illness

Oral lesions are variable; some isolates cause major oral lesions while in infections by other isolates, there is no oral lesion. Oral lesions start as small, grey foci that may coalesce. The grey (necrotic) epithelium then sloughs off, leaving a red erosion.

Photographs of Clinical Signs of Acute RP

13 The animal is drooling.

14 There is a mild hyperemia of the eyelids, mild conjunctivitis, a small amount of exudate in the medial canthus, and wet hair beneath the medial canthus.

15 Reddened, blunted, conical papillae at the commissure of the lips.

16 Necrosis and erosion of the gum above the dental pad and inside the upper lip.

17 Necrosis and erosions on the lower lip, dental pad, commissure of the lips, and hard palate.

18 Erosions in the inside of the upper lip.

Clinical Signs of the Mild or Transient Form of RP

19 The mild form is seen in infections by less virulent isolates or in an area with endemic RP where calves may have some colostral immunity. These animals may have many signs, such as fever, anorexia, nasal and lacrimal discharges, depression, and diarrhea. But these are often transient or so mild as to be missed, and the animal recovers.

Gross Lesions in Acute or Classic RP

20 RPV has an affinity for epithelial and lymphoid tissues. The primary lesions are in the oral cavity and small and large intestines.

Mouth—Lesions occur on the gums, lips, hard and soft palate, cheeks, and base of the tongue. Early lesions are grey, necrotic, pinhead-size areas that later coalesce and erode, leaving red areas.

Esophagus—Brownish necrotic or eroded areas occur.

Rumen and reticulum—Lesions are rare.

Omasum—Erosions and hemorrhages are rare.

Abomasum—Congestion and edema occur.

Small intestine—In the jejunum, there is necrosis or erosion of the Peyer's patches. In the ileum, there is necrosis or erosions over the lymphoid area. When the intestines are opened at necropsy, ingesta are found adhering to the mucosa, indicating areas of necrotized epithelium.

Caecum and colon—The wall may be edematous, and there may be blood in the lumen and blood clots on the mucosa. Lesions are usually more severe in the upper colon; edema of the wall, necrotic foci in the mucosa, and congestion may be present. The lesions may be accentuated at the ceco–colic junction. Farther down the colon, the colonic ridges may be congested (tiger striping); this probably results from tenesmus.

The severity of lesions in the intestines varies, depending on the infecting RPV isolate.

Lymph nodes—These are generally swollen and edematous.

Gall bladder—There may be petechial to ecchymotic hemorrhages in the gall bladder.

Lung—There may be emphysema, congestion, and areas of pneumonia.

Photographs of Gross Lesions

21 Grey epithelium and small linear erosions at base of the tongue. Frequently this is the only oral lesion in RP infected animals.

22 Two necrotic Peyer's patches. The necrosis in the grey Peyer's patch is more advanced than in the other. In both, the epithelium is necrotic but has not yet sloughed off.

23 This Peyer's patch has lost the epithelium, and there are blood clots on the surface.

24 An eroded Peyer's patch. The white foci in the eroded Peyer's patch is necrotic material in crypts.

25 Lower ileum. This whole area is underlaid by lymphoid tissue. Note all the erosions in the epithelium and smallamount of exudate on the surface.

26 Closeup of erosions in the lower ileum.

27 Cecum at the ileocecal valve. All the darker red areas are erosions of the cecal mucosa. The mucosa is covered by a layer of fibrin. You can see the imprints of the erosions on reflected areas of fibrin.

28 Colon—Severe area of hemorrhage.

29 Rectum—Hyperemia of the longitudinal folds (tiger striping).

30 Closer view of 29.

31 In the cortex of the lymph node, there are many small white foci; these are necrotic follicles.

32 Multiple hemorrhages in the mucosa of the gall bladder.

Microscopic Lesions

33 Epithelial necrosis starts in the stratum spinosum just above the basal layer and extends to the surface. Occasionally there may be syncytial cell formation in this area. When the necrotic epithelium sloughs off, an erosion is formed—erosion because the basal layer is still intact. Tonsilar crypts are a good place to find syncytial cells.

Lymph nodes—There is disappearance of mature lymphocytes and necrosis of lymph follicles. There may be syncytial cells in the necrotic follicles.

Spleen—There is necrosis of lymph follicles.

Photomicrographs

34 Necrosis, inflammatory cell infiltrate, and syncytial cells are found in the oral epithelium. The syncytial cells are a significant feature for making a diagnosis of RP.

35 Mesenteric lymph node—There is lymphocyte depletion, but the significant lesion is that the cells in the germinal center of the follicle are very enlarged, and there are syncytial cells.

36 Mesenteric lymph node—There is necrosis of cells in the germinal center and one syncytial cell.

Morbidity and Mortality

37 In the classic form of RP, morbidity and mortality in a naive population of cattle can be very high.

Diagnosis

Field Diagnosis

38 RP should be considered whenever there is a rapidly spreading, acute, febrile disease—accompanied by the aforementioned clinical signs and lesions of RP—in **all ages** of cattle. The **all ages** is important because this will be one of the major features distinguishing RP from bovine viral diarrhea—a mucosal disease—which predominantly affects animals between 4 and 24 months of age.

Specimens for Laboratory

39 The viral titer drops when the fever falls and diarrhea starts, so it is preferable to collect specimens from animals with a high fever and oral lesions. The following samples should be collected from live animals:

- Blood in EDTA or heparin
- Blood for serum
- Swabs containing lacrimal fluid
- Necrotic tissue from the oral cavity
- Aspiration biopsies of superficial lymph nodes

For the best specimens, a febrile animal should be slaughtered and specimens should be collected. If this is not possible, specimens should be collected

from moribund animals. Collect the abovementioned blood samples and sections of:

- Spleen
- Lymph nodes
- Tonsil

Transport the specimens to the laboratory on wet ice, **not frozen.**

A complete set of tissues, including sections of all lesions, should be collected in 10-percent formalin.

Laboratory Diagnosis

40 **Virus Isolation**—To confirm the initial diagnosis in a free area, one must isolate and identify the virus. Virus isolation can be done in the lab using primary calf kidney cells or Vero cells, or it can be done by inoculating live cattle. Viral isolates are identified in cell culture using virus neutralization, direct immunofluorescent staining, or direct immunoperoxidase staining.

RP antigen in spleen, lymph node, necrotic gum epithelium, and even lacrimal swabs can be detected by an immunodiffusion test or counterimmunoelectrophoresis test.

Serology—RP antibody can be detected by virus neutralization or an electroimmunosorbent assay (ELISA).

Vaccination

41 Several types of RP vaccine have been used:

- Lapinized (China and Korea)
- Avianized–lapinized (Korea)
- Goat-adapted (India)

- Cell-culture-adapted (Africa, Middle East, India)

- An experimental vaccinia-vectored vaccine containing the F and H genes of RPV.

The two RP vaccines most commonly used today are the goat-adapted and cell-culture-adapted vaccines.

The goat-adapted vaccine is only partially attenuated; it will cause disease in animals with low innate resistance or concurrent latent disease and kills sheep and goats.

The cell-culture-attenuated vaccine was developed by Plowright in Kenya in the 1960's. It is a safe vaccine for many species and produces lifelong immunity in cattle. Animals challenge-inoculated 7 years after vaccination were found to be protected. In areas where RP is endemic and cattle have been vaccinated, colostral immunity will interfere with the vaccination of calves up to 11 to 12 months of age. Because the duration of colostral immunity is variable, the recommendation is to vaccinate calves annually for 3 years.

One of the biggest problems with the cell-culture-adapted vaccine has been stability. The lyophilized virus has to be kept cold (cold chain) until used. The remoteness of vaccination sites has made meeting this requirement very expensive. Because of uncertainty about whether the vaccine being used is viable, in some areas of Africa, it has been the policy to vaccinate animals every year in hopes that one of the vaccinations will immunize them.

Researchers at Plum Island in the early 1990's greatly increased the stability of the lyophilized vaccine by modifying the stabilizers and lyophilization process. This change in processing is now being used in some production facilities in Africa.

In experiments, the vaccinia-vectored RP vaccine has been found to protect cattle challenge-inoculated with RPV. This vaccine is undergoing field testing. Vaccinia-vectored RP vaccine could be particularly useful in an eradication program because animals immunized with it can be differentiated serologically from animals having antibody induced by live virus. The vaccinia-vectored vaccine would enable a country toward the end of an eradication program to maintain herd immunity to RP without using a live RP virus.

Control or Eradication

42 Countries and areas free of RP should prohibit unrestricted movement of RP-susceptible animals and uncooked meat products from areas infected by RP or areas where RP vaccination is practiced. Because recovered animals are not carriers and good serological techniques exist, zoo ruminants and swine can be imported with proper quarantine and testing. If an outbreak occurs, the area should be quarantined, infected and exposed animals should be slaughtered and buried or burned, and ring vaccination should be considered.

43 Experiments have shown that RPV will not be transmitted by bovine embryo transfer if the embryos have been processed by the technique recommended by the International Embryo Transfer Society and the International Office of Epizootics.

High-risk countries—countries trading with or geographically close to infected countries—can protect themselves by having all susceptible animals vaccinated before they enter the country and/or vaccinating the national herd. If an outbreak occurs, the area should be quarantined and ringvaccinated.

Countries where RP is endemic should vaccinate the national herd. Because of the uncertainty about the potency of the vaccine, the recommendation is to vaccinate the herd annually for at least 4 years, with annual vaccination of calves. Foci of infection should be quarantined and stamped out. Wildlife and sheep and goats should be monitored serologically. Serological monitoring of sheep and goats could be complicated by using RP vaccine to protect against peste des petits ruminants.

Peste des Petits Ruminants

Definition

1 Peste des petits ruminants (PPR) is an acute to subacute contagious viral disease of goats and sheep characterized by erosive stomatitis, conjunctivitis, diarrhea, and pneumonia.

Synonyms for PPR include pest of small ruminants, stomatitis–pneumoenteritis complex or syndrome, pseudorinderpest of small ruminants, and kata (means catarrh).

Etiology

2 PPR virus (PPRV) is a single-stranded RNA virus in the family Paramyxoviridae, genus *Morbillivirus*. Rinderpest virus and PPRV are closely related.

PPRV can be differentiated from rinderpest virus by the virus neutralization test. Cell cultures of PPRV incubated at 40 °C remain pathogenic; cultures incubated at 37 °C will become attenuated.

Effective Disinfectants

Strong acid and alkaline disinfectants and many common disinfectants, particularly the iodophore and chlorine dioxide disinfectants, are effective.

History

3 PPR was first recognized as a distinct disease entity in the Ivory Coast in 1942. The viral agent was isolated in 1956 and was thought to be a rinderpest virus that had adapted to sheep and goats.

Host Range

4 Goats and sheep are the major species affected by PPR; goats are more severely affected than sheep. Cattle and pigs can have a subclinical infection but do not shed virus. PPR has occurred in these wild ungulates in captivity: gazelle, ibex, and Laristan sheep. White-tailed deer infected experimentally suffer a fulminant form of the disease.

Geographic Distribution

5 PPR is present in Africa in the area between the Sahara Desert and the equator, in the Middle East, and in the Indian subcontinent.

Transmission and Epidemiology

6 Transmission of PPR is similar to transmission of rinderpest—PPR spreads rapidly in goats and sheep in close contact. Intranasal–oral transmission occurs through the inhalation of droplets produced by sneezing and coughing and ingestion of contaminated food and water. PPRV is present in nasal, pharyngeal, and conjunctival secretions and in saliva, urine, and feces. The virus is in nasal secretions 3 days after infection. There is no carrier state.

In endemic areas, PPRV is always present, but the incidence of the disease is cyclic. After an outbreak, antibody levels are high, and the virus dies out in many herds. A susceptible population develops, and when PPRV is reintroduced, an epizootic with almost 100-percent mortality may occur.

Animals under 6 months of age succumb to diarrhea; older animals surviving the diarrhea phase of PPR will develop pneumonia. Goats are usually more susceptible than sheep. The severity of disease and associated mortality are increased in animals in a poor state of nutrition or suffering from concurrent parasitic or bacterial disease.

Incubation Period

7 The incubation period for PPR is usually 4 to 5 days.

Pathogenesis

8 The portal of entry for PPR is probably the epithelium of the upper respiratory tract and/or tonsil.

PPRV replicates in the local lymph nodes. The virus spreads via the blood to other lymph nodes, conjunctivae, oral cavity, and the mucosa of the intestinal and respiratory tracts.

Viremia and shedding occur 1 to 2 days before the first clinical sign, fever.

Destruction of the lymphoid tissue causes immunosuppression and may result in recrudescence of hemoparasitic and other secondary infections, such as bronchopneumonia.

Clinical Signs

9

The first clinical sign of PPR is fever of 104 to 106 °F (40 to 41.1 °C). Anorexia and general signs of illness follow.

Nasal discharge starts out serous but may progress to a mucopurulent exudate that occludes the nostrils. The animal will have respiratory distress and sneeze.

Ocular discharge starts out serous but can progress to a mucopurulent exudate that mats the eyelids closed.

The epithelium around the nostrils and lips and in the mouth will become necrotic and slough, forming erosions. Oral lesions—on the lips, gums, buccal papillae, tongue, and hard and soft palate—appear 7 to 8 days after infection.

A profuse to watery diarrhea starts about the time the oral lesions appear.

Coughing and sneezing commonly occur because of pneumonia.

The animal becomes dehydrated and emaciated, and death usually occurs about 5 to 10 days after the onset of clinical signs.

Animals may recover from the acute disease, but some will be unthrifty and eventually die of pneumonia.

Photographs of Clinical Signs

10

Goat is very sick—depressed.

11

Extensive necrosis of epithelium around the eyes, nose, and mouth.

12 Excessive lacrimation—bilateral wet hair under medial canthus and nasal exudate.

13 Necrosis and erosions on the lips.

Gross Lesions

14 In acute PPR, the carcass is dehydrated and soiled.

Nostrils are encrusted with a mucopurulent exudate, and the epithelium is necrotic and eroded. The animal has severe conjunctivitis, necrosis of epithelium at the edges of the eyelids, and a mucopurulent exudate matting the eyelids closed. Lips are hyperemic. Oral lesions vary from a few erosions on the soft palate to extensive necrosis and erosions on the inside of the lips, commissures of the cheeks, gums, tongue, and hard and soft palate.

Lesions in the small intestine are usually not remarkable. According to the literature, lesions similar to those in rinderpest sometimes occur. Patches of congestion (atelectasis) in the lung and consolidation of the apical and cardiac lobes occur. Lymph nodes are enlarged and edematous. The spleen may be slightly enlarged and congested.

Photographs of Gross Lesions

15 The white areas on the base of the tongue, pharynx, and soft palate are areas of necrotic epithelium.

16 Pneumonia and atelectasis.

17 Pneumonia—Note that pneumonic lesions occur throughout the lung.

Microscopic Lesions

18 In the necrotic and eroded oral epithelium, there may be cytoplasmic and intranuclear eosinophilic inclusions and an occasional syncytial cell.

In the lung there may be giant-cell pneumonia and intranuclear and intracytoplasmic inclusions.

In the spleen, lymph nodes, and tonsils, there will be necrosis of lymphocytes, and there may be syncytial cells and inclusions.

Photomicrographs

19 There are giant cells in the bronchioles and some alveoli and neutrophils in alveoli. This giant-cell pneumonia is similar to that seen in pneumonias caused by other morbilliviruses, specifically measles virus in primates and canine distemper virus in dogs.

20 Giant-cell pneumonia; note the very large syncytial cells.

21 Another area of lung tissue showing giant-cell pneumonia note the very large syncytial cells.

22 Oral mucosa; cells in the stratum spinosum are enlarged and separated; the squamous cell layer is one cell thick. There are many sloughed-off cells above the mucosa; some of the larger round cells are suggestive of syncytial cells.

Morbidity and Mortality

23 In a susceptible herd, morbidity may approach 100 percent. Mortality in young goats (under 8 months old) can be 10 percent to 95 percent; mortality in young sheep is usually lower than in goats. Older animals frequently recover.

Diagnosis

Field Diagnosis

24 A presumptive diagnosis of PPR should be made when there is an epizootic in sheep and goats accompanied by mortality and suspicious signs and lesions of PPR.

Specimens for Laboratory

25 The following samples should be submitted:

- Blood in EDTA or heparin
- Blood for serum
- Spleen
- Lung
- Lymph nodes
- Tonsil
- Swabs containing lacrimal fluid
- Necrotic tissue from the oral cavity

The above samples should be transported to the laboratory on wet ice, **not frozen.**

A complete set of tissues including sections of all lesions should be collected in 10-percent formalin.

When possible, also submit paired serums from survivors. In PPR-free areas, serums from convalescent animals are also of value.

Laboratory Diagnosis

26 To confirm the initial diagnosis in a free area, the virus has to isolated and identified.

Virus Isolation—Virus isolation can be done in cell culture or by animal inoculation. Antigen can be detected by agar gel precipitation and by counterimmunoelectrophoresis.

Serology—Antibody can be detected by virus neutralization, agar gel precipitation, counterimmunoelectrophoresis, or indirect enzyme-linked immunosorbent assay (ELISA).

Pathology—Histologic lesions can be quite specific for PPR.

Vaccination

27 Rinderpest vaccine will immunize against PPR. Protection will last at least 12 months.

PPR vaccines are being developed.

Control or Eradication

28 & 29 Countries and areas free of PPR should prohibit unrestricted movement of PPR-susceptible animals from areas infected by PPR or from areas where PPR vaccination is practiced. Because recovered animals are not carriers and there are good serological techniques for detecting antibody, ruminants and swine for zoos can be imported with proper quarantine and testing. If the disease is introduced, it can be eradicated by quarantine, slaughter and disposal of infected animals, and decontamination of the affected premises.

High-risk countries—countries trading with or geographically close to infected countries—can protect themselves by having all susceptible animals vaccinated before they enter the country and/or vaccinating the national herd. If an outbreak occurs, the area should be quarantined and ring-vaccinated.

In areas where PPR is endemic, animals should be vaccinated. If PPR occurs, treatment should be directed at controlling secondary diseases, particularly pneumonia.